THE DEAD ARM DISEASE OF GRAPES IN ONTARIO.

A PRELIMINARY STUDY.

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The Dead Arm disease of the grape vine has, within recent years, become the most serious one with which the grape growers of Ontario have to contend. This, together with the fact that our knowledge of the disease is imperfect and the control measures used inadequate, led me to take up its study in June, 1926. As circumstances have compelled me to break off the work permanently, I have felt it advisable to publish the results already obtained although I realize fully that these are only of a preliminary character.*

The scanty literature on the subject up to 1914 has been carefully reviewed by Reddick, whose two papers *(1, 2)* contain most of the information on the disease as it occurs in America. I shall attempt no review here but would refer readers to Reddick's last paper. Since 1914 there has been, as far as I have been able to ascertain, only one paper on the subject, that by Hiura *(3, 4)*.† This author published in 1924 the description of a disease of the grape occurring on the island of Sapporo, Japan, which leaves hardly any doubt that he has been dealing with the same disease. His paper will be referred to later.

Distribution

Shear *(5)* gives the distribution of the disease as practically the whole of the grape growing area of the Eastern United States. The present studies have been confined to the Niagara peninsula where the disease is general. During the summer of 1927 Mr. D. R. Sands of the Ontario Agricultural College, Guelph, sent me material collected in Kent County, Ontario, which showed that the disease is present in the southwestern part of the province also. According to Palmer and van Haarlem's *(6)* recent bulletin these two regions contain most of the commercial vineyards of the province.

Varieties Attacked

Reddick *(2)* has noted that the disease attacks every variety grown commercially in New York State. This is true of the Niagara peninsula also, for I have yet to see a variety quite free from the disease. There is, however, a very marked difference in the severity and extent of attack on the different varieties. Reddick reports that Delaware seems to have a certain amount of resistance and my observations in Ontario corroborate this. However, it is not the only variety which appears to show resistance in our area.

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*The investigations were carried out during the summer of 1926 in the Dominion Field Laboratory of Plant Pathology, St. Catharines, and since then in the Botany Department, University of Toronto and a field laboratory on the Provincial Horticultural Experiment Station, Vineland, Ontario. I have to thank Dr. G. H. Berkeley, in charge of the former laboratory, and his staff, for their very generous help and cooperation, and Mr. E. F. Palmer, Director of the Provincial Horticultural Experiment Station, for the excellent facilities for study provided at Vineland. During the past year I have been assisted in this work by Mr. R. F. V. Cooper, to whom my thanks are also due.

† I have to thank Dr. G. H. Berkeley for bringing the first of these papers to my attention.

Table 1 gives the results of a careful examination of sixteen varieties growing in neighbouring rows on the Horticultural Experiment Station, Vineland. The examination involved a careful inspection of all the above ground parts of the vines, the stems and arms having the old dead bark carefully stripped off before examination. In the table the varieties are arranged according to the percentage of vines showing the characteristic leaf symptoms which, to grape growers, furnish the first evidence of the disease. As will be shown later, stem and arm lesions are to be found some considerable time before leaf symptoms appear and their occurrence has also been noted in the table.

TABLE 1.—*Occurrence of Dead Arm on sixteen varieties of grapes grown on the Horticultural Experiment Station, Vineland. Age of vines, 16 years.*

Variety	Total No. of vines	Vines showing leaf or arm symptoms	Vines showing lesions on stem or arm but no other symptoms.	Total No. of vines attacked.	Percentage of vines showing leaf or arm symptoms.	Percentage of vines attacked.
1. Concord	28	11	3	14	39.2	50
2. Worden	27	10	2	12	37	44
3. Moore's early	23	7	0	7	30.4	30.4
4. Campbell early	22	6	4	10	27.2	45.4
5. Lindley	28	7	6	13	25	46.4
6. Diamond	27	6	4	10	22.2	37.
7. Agawam	28	6	6	12	21.4	42.8
8. Herbert	28	3	1	4	10.7	14.3
9. Massasoit	28	5	3	8	18.5	29.6
10. Brighton	28	3	1	4	10.7	14.3
11. Pocklington	28	3	1	4	10.7	14.3
12. Wilder	28	3	0	3	10.7	10.7
13. Salem	28	1	11	12	3.6	42.8
14. Delaware	28	1	2	3	3.6	10.7
15. Vergennes	28	1	1	2	3.6	7.2
16. Niagara	28	1	0	1	3.6	3.6

The second column of the table gives the actual number of vines growing in each row. Where this number is less than 28, the difference represents the number of vacancies, which almost certainly have resulted from the killing of vines by the disease. Four varieties show these vacancies, namely, Worden (1), Moore's Early (5), Campbell Early (6), and Diamond (1). If we add these numbers to those given in column 5, it brings the totals up nearly to the figures given for Concord. We may therefore say, as far as our figures indicate, that the first seven varieties given in the table, viz. Concord, Worden, Moore's Early, Campbell Early, Lindley, Diamond and Agawam are all highly susceptible to the disease, and about equally so. The remaining nine varieties seem to be much less susceptible with the exception of Salem, which, while it showed only one vine with leaf symptoms, had an additional eleven showing stem lesions.

The last three varieties showed a very small percentage of infection. Of these the last, Niagara, is our most important white grape and the fact that it shows so little attack is a matter of considerable importance.

The severity of attack as evidenced by the removal of vines or arms does not parallel the percentage of attack in the different varieties. Thus the figures would seem to indicate that Moore's Early and Campbell Early, when once attacked, succumb sooner than do the other varieties. It might be suspected that lack of vegetative vigour is directly correlated with severity of attack but this can hardly be the case as Diamond is certainly not as vigorous a variety under the conditions existing at Vineland as either Moore's Early or Campbell Early. It should be noted also that the percentage of attack on the varieties apparently more susceptible, cannot be accounted for by any greater opportunity of infection as the rows are not arranged in the vineyard in the order given in the table. As an example, Niagara with one vine attacked out of 28 lies next to Worden with 12 attacked out of 27. The varietal rows examined form a solid block and are on apparently uniform land so the chances of infection should be fairly equal throughout.

The number of vines examined belonging to any one variety is admittedly too small to allow for the drawing of definite conclusions as to susceptibility. However, it is impossible in the Niagara peninsula to find large numbers of vines of any considerable number of varieties growing under similar conditions, so it is very doubtful if more accurate comparative data can be obtained than those given above.

One of the most striking facts revealed by the figures in this table is the quite unexpectedly high percentage of attack on nearly half of the varieties examined, among which are some of those most extensively grown. Thus Concord, our leading commercial variety, stands at the head of the list with 50 per cent of the vines attacked. It should be stated at once that where large blocks of a single variety have been examined such high infestation has rarely been found, but it has nevertheless been much higher than is generally supposed.

To obtain data on the extent of the disease in the Niagara peninsula, extensive field studies involving the examination of about 2,500 vines were made during the early summer of 1927. The vineyards examined were among the best to be found in their particular areas, and were all in an excellent state of cultivation. As a matter of fact, I have visited a number of vineyards where the infestation was much greater. The following tables summarize the results of this examination:

Vineyard No. 1.

This vineyard is situated in the neighbourhood of St. Catharines and forms a part of 60 acres of grapes consisting largely of the Concord variety, with a few Wordens mixed in. The portion examined lies in a block which is probably the most severely attacked of the whole area but the disease is severe in other areas also. Table 2 presents an analysis of the field examination.

TABLE 2. *Analysis of disease in eight rows of vineyard No. 1—Variety chiefly Concord, with some Worden mixed. Age of vineyard, 15 years.*

	Row 1	Row 2	Row 3	Row 4	Row 5	Row 6	Row 7	Row 8	Total
Healthy vines	85	45	58	43	46	50	46	51	424
Replacements	2	28	22	22	26	26	24	23	173
Vines showing leaf symptoms and lesions	6	10	11	24	12	17	18	13	111
Vines showing lesions only	2	10	2	5	12	6	8	3	48
Vacancies	1	2	2	1	0	0	1	1	8
Total	96	95	95	95	96	96	96	95	764

Percentage of adult vines showing leaf symptoms and lesions — 19.1.

Percentage of adult vines attacked as evidenced by lesions on stem or arms — 27.3.

The fact that in the rows examined there were some Worden vines mixed with the Concords probably has had no effect upon the results, as these two varieties appear to be about equally susceptible. It should be noted here that the examination of these vines involved the stripping of old bark from stems and arms and a careful search for lesions. These lesions or cankers will be described later and are to be looked upon as the seat of the disease in the vast majority of cases. They are, of course, usually overlooked by the grape grower.

The first line in the table shows the number of healthy vines in full bearing per row, the total in the eight rows being 424 against a total possibility of 764. While, therefore, the percentage of adult vines attacked is 27.1, the total percentage of vines killed or threatened by the disease is 44.5. In arriving at this figure I am assuming that all the vacancies and replacements have been due to the disease. This assumption is, I believe, largely justified. The only other probable cause of death is cultivation injury and an examination of many acres in the vineyard during the summer of 1926 showed practically no evidence of this. Winter killing could hardly have been a factor in this vineyard.

Under replacements I have included all young vines which have not yet nearly reached full bearing. In some cases these are from actual replantings. In others they are from suckers brought up from a stub cut at ground level. As cultivation had, for the most part, buried these stubs and as the stumping had been done as a result of the disease, no attempt was made to keep the two kinds of replacement separate. A determined effort is being made in this vineyard to bring the disease under control, which partly accounts for the high number of replacements.

Vineyard No. 2.

This vineyard is situated at Vineland. It consists chiefly of Concords and has been planted fifteen years. Practically no vines have been removed so no record was kept of replacements or vacancies. The examination was made on May 31, 1927, and subsequent days, by which time typical leaf symptoms had not developed. The record, therefore, is based solely on the occurrence of lesions or cankers on the stem and all doubtful or incipient lesions were excluded from the estimate.

TABLE 3.—*Results of inspection for Dead Arm in vineyard No. 2. Variety Concord, Age 15 years.*

Number of vines examined — 236.
Number of vines showing definite stem lesions — 41.
Percentage of infection —17.3.

Vineyard No. 3.

This vineyard is situated at Winona and consists almost exclusively of the Agawam variety. In the examination only those vines showing typical leaf symptoms were considered as attacked, the actual presence of the disease on such vines being verified by the occurrence of stem lesions.

The vineyard contains fifteen rows and of these eight were examined, four on the east side and four on the west. As two of the eastern rows were short ones, approximately half the vineyard was examined. The examination was made on June 30, 1927, with the results given in Table 4.

TABLE 4.—*Results of inspection for Dead Arm in vineyard No. 3. Variety Agawam. Age 15 years.*

	Row 1	Row 2	Row 3	Row 4	Row 12	Row 13	Row 14	Row 15	Total
Healthy vines	44	37	77	71	71	79	71	74	524
Vines showing leaf symptoms	7	13	14	17	7	11	17	13	99
Vacancies	3	7	4	6	21	8	11	8	68
Total	54	57	95	94	99	98	99	95	691

Percentage of disease on adult vines present — 15.9.
Percentage of disease and vacancies on total spaces in rows — 24.0.

As will be noted from the table, there had been no recent replacements in this vineyard, all the vines present being those apparently of full bearing age. The number of vacancies was correspondingly high. I was assured by the foreman in charge that most of the removals had occurred during the past two or three years and all the evidence indicated that these had been due to death from Dead Arm. The ratio of diseased vines plus vacancies to the total number of spaces in the rows examined must therefore give us a more accurate idea of the damage done by the disease than the ratio of diseased vines to bearing vines. As will be noted, while the disease does not seem to be so severe as in vineyard No. 1, it is still quite serious. As only vines exhibiting leaf symptoms are shown as diseased, the percentages given are probably much below the actual.

Vineyard No. 4.

This vineyard is at Vineland and the portion examined is a block consisting almost exclusively of Moore's Early vines. It is fifteen years old. Examination was carried out on July 9, 1927, and here again, owing to lack of time, attention was confined to those vines which showed leaf symptoms. The results are summarized in Table 5.

TABLE 5.—*Results of inspection for Dead Arm in vineyard No. 4. Variety Moore's Early. Age 15 years.*

Total number of healthy vines excluding those apparently of other varieties	559
Total number of vines showing Dead Arm leaf symptoms	69
Winter killed vines (no lesions)	4
Replacements (non-bearing vines)	45
Vacancies	6
Other varieties	8
Total	691

Percentage of bearing vines attacked — 11.
Percentage of attacked vines, vacancies and replacements to total spaces—17.6.

In the same vineyard there were two rows of Niagara vines, 12 years old, which were examined. Of the eighty-eight vines in these rows only two showed leaf symptoms accompanied by stem lesions. There was, in addition, one vacancy which may or may not have been due to this disease.

Vineyard No. 5.

This vineyard is also located at Vineland and contains one of the largest blocks of Niagara vines in the neighbourhood. It was to gain further information as to the susceptibility of this variety that examinations were made here on July 12, 1927. The six rows of Niagara vines examined were 16 or 17 years old and contained a total of 462 vines. Attention was confined to leaf symptoms owing to lack of time. While a certain number of the vines showed chlorotic leaf symptoms, these were not typical of Dead Arm nor were there any stem lesions accompanying them. It seems, then, that this block is almost or quite free from disease. A hurried examination of a number of rows of Concord vines directly adjoining the Niagara rows showed typical Dead Arm leaf symptoms accompanied by stem lesions. Counts were not made owing to want of time but the percentage of infestation was lower than in the other Concord blocks examined.

The above statements, along with Table 1, give the results of an examination of more than 3,000 grape vines, including all the varieties grown commercially in the province. Where it has been possible to examine large numbers of any particular variety in a solid block, the figures have roughly corroborated those obtained from an examination of a smaller number grown in a varietal vineyard. The field studies have also been extended over a fairly wide area in the Niagara peninsula and can, I think, be taken to give a fair picture of the incidence of the disease in commercial vineyards. Undoubtedly there are vineyards showing a higher infestation than those examined, while there are as certainly others in which the disease is not so serious. The figures show that some of our most important grape varieties such as Concord, Worden, Moore's Early and Agawam are decidedly susceptible while, on the other hand, our most important white variety, Niagara, seems to be highly resistant.

It will be noted that all the vineyards examined are over ten, and under twenty, years old. There are younger vines in most of these vineyards in the form of replacements. These have, in almost every case, shown no signs of the disease. The same freedom from disease has been noted in young vineyards which have been visited in the course of field studies. In fact, during the two years' field work, I have seen no vine under five years of age with definite leaf symptoms. One stem lesion was found around a pruned stub on the stem of a vine three or four years of age, while a very few vines of seven or eight years have shown leaf symptoms. The great majority of vines showing leaf symptoms have, however, been over ten years of age. The belief that young vines do not show the disease is widespread among grape growers and I believe this view is well founded. Possible reasons for this interesting state of affairs will be considered later. As regards older vineyards, while I have made no definite estimates of the percentage of diseased vines, observations indicate that the disease increases in seriousness with the increasing age of the vineyard.

While the figures given above show us the degree of infestation in vineyards between ten and twenty years of age, they do not enable us to estimate the percentage of vines attacked each year. In order to make a rough estimate of this is would be necessary to know what period, on the average, elapses between inoculation and death of the vine. An examination of stem lesions on dead vines or on those showing very advanced symptoms shows that they are frequently four or five years old. I think we may estimate the period from inoculation to death as on the average about five years. With infections running from 10 per cent to 50 per cent on susceptible varieties, that would give us an annual incidence of from 2 to 10 per cent. In the case of Niagara, Delaware and a few less important varieties, the annual incidence is apparently almost negligible.

Effect of the Disease on Yield.

The impression seems to be fairly wide-spread among grape growers that Dead Arm, while killing off a few vines here and there, has no serious effect upon the yield. This belief is apparently based largely on the fact that the vines seem to recover during the latter half of the growing season, a matter which will be discussed fully later in this paper. It may also be partly due to the fact that other chlorotic leaf symptoms are, at times, confused with the typical leaf symptoms of Dead Arm and, as is well known, leaf chlorosis quite frequently disappears during July and August.

An attempt has been made during the harvesting season of 1927 to get comparative yield figures from diseased and healthy vines in some of the vineyards which were examined early in the summer. The work of keeping yields from vines growing close together in a row separate is a burdensome one and as the harvest in commercial vineyards has to be hurried through it has not been possible to do as much in this regard as is desirable. However, a beginning has been made and it is hoped that the figures obtained may be supplemented in subsequent years.

It was early recognized that in order to obtain figures of any value it would be necessary to keep yield records from a large number of vines. An analysis of figures kindly placed in my hands by Mr. van Haarlem of the Horticultural Experiment Station, Vineland, showed such a large variation in yield from vine to vine among healthy vines as to make the use of figures from small numbers quite valueless for this purpose.

Yields were recorded in three vineyards, one at Vineland, one at Winona and one at St. Catharines*.

The harvest records from the vineyard at Vineland gave nothing which would enable us to judge of the effect of disease on yield; in fact the average yield from diseased vines was greater than that from the healthy ones. This was, I think, partly due to the fact that the record was kept from too small a number of vines (a total of 118 vines of which 38 were diseased) and partly to the fact that the vines designated as diseased included all those showing stem lesions. As already noted, the leaf symptoms were not visible at the time these vines were marked and as a matter of fact they were not strongly marked in this vineyard. As the figures given below suggest, an appreciable reduction in yield apparently does not set in till after definite leaf symptoms have appeared.

As the yield figures obtained from this vineyard cannot be considered as significant they are not included. However, they do indicate that the variation in yield from vine to vine is very great. Unfortunately the conditions of harvest were such as to prevent our keeping the yields of the individual vines separate so we have no way of judging the extent of experimental error due to this variation. In the other two vineyards yield figures from a much larger number of vines were kept. They are therefore given below. It will be realized that these figures are preliminary only and must be supplemented by fuller ones from subsequent harvests if we are to judge at all accurately the effect of the disease upon yield.

Comparison of Yield in Vineyard No. 1.

In this vineyard records of six out of the eight rows examined earlier in the season were kept. In two of these a comparison was made between the yield of vines showing conspicuous stem lesions with that from the remaining adult vines of the rows, some of which showed small stem lesions. Our numbers here therefore, do not correspond with numbers given in Table 2. Table 6 gives the results:—

Table 6.—*Yield from healthy and diseased vines in Vineyard No. 1. Variety Concord.*

	Healthy vines			Diseased vines		
Row	No. of vines	Total yield in lbs.	Av. yield in lbs.	No. of vines	Total yield in lbs.	Av. yield in lbs.
1	35	437	12.5	20	258	12.9
2	43	555	12.8	13	150	11.5
Total	78	992	12.7	33	408	12.4

The figures in Table 6 show no significant difference in yield between the so-called healthy vines and the diseased ones. It must be remembered, however, that some of the vines included under "healthy" were really diseased. The "diseased" vines included many which had not yet shown leaf symptoms and so would have been looked upon as healthy by grape growers.

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*Thanks are due to Messrs. E. D. Smith & Sons, Winona, The Ontario Grape Growing and Wine Manufacturing Co., Ltd., St. Catharines, and Mr. G. W. Philbrick of Vineland for their cooperation in this work.

In any case, these figures suggest that there is no perceptible reduction in yield in diseased vines before the leaf symptom stage has appeared.

In the other four rows the yield from adult vines which had shown marked leaf symptoms and which therefore represented vines which would be generally recognized as diseased, was kept separate. The results are shown in Table 7.

Table 7. *Comparison of yield from vines showing leaf symptoms with those from vines showing no leaf symptoms. Variety Concord.*

	Vines showing no Leaf Symptoms			Vines showing Leaf Symptoms		
Row	No. of vines	Total yield in lbs.	Av. yield in lbs.	No. of vines	Total yield in lbs.	Av. yield in lbs.
1	53	695	14.0	12	133	11.6
2	53	689	12.9	11	139	12.6
3	49	630	12.9	17	247	14.5
4	54	775	14.4	15	127	8.4
Total	209	2789	13.35	55	646	11.76

In this case, the reduction in yield indicated is 12.1 per cent, a figure which may possibly be significant although even here there is one row in which the diseased vines yielded more than the healthy.

These figures do not, of course, show the real loss of yield, for the reduction in yield on replacements and due to vacancies is not considered. In these four rows there were fifty-five young vines which produced an average crop of only 2.7 lbs. per vine. In addition, there were sixty-seven very young vines and vacancies from which no crop at all was harvested. If we add the yield from the young vines to the yields as found in Table 7 we obtain a total yield of 3,584 lbs. from an area which should contain 383 vines or an average yield of 9.35 lbs. per vine. If we compare this with the average yield of 13.35 lbs. per vine given for the so-called healthy vines in the above table, we find a reduction of almost exactly 30 per cent. This figure shows what a really serious effect the disease may have upon the production of a vineyard.

Yield in Vineyard No. 3.

In vineyard No. 3, yield records were kept from a number of diseased vines in five out of the eight rows examined in July. In this case it was impossible to keep a record according to rows. The crop from vines marked as having shown pronounced leaf symptoms was picked and weighed separately and the total compared with the total from the remaining vines in these rows. As there had been no recent replacements in this vineyard the results were not complicated by the presence of young vines. However, there were quite a number of vacancies. An analysis gave the following figures:

Vines showing no leaf symptoms or only slight ones....	292
Vines showing marked leaf symptoms....	60
Vacancies	29
Total	381

The 292 "healthy" vines gave a net yield of 4,544 lbs. or an average of 15.6 lbs. per vine. The 60 diseased vines gave a net yield of 747 lbs. or an average of 12.45 lbs. per vine. There was a reduction of 3.15 lbs. per vine or 20.2 per cent as compared with that from the "healthy" vines. If we take into account the vacancies in these rows, we find an indicated reduction in yield of 653 lbs. of grapes or almost exactly 10 per cent on the whole area. In this case, owing to the smaller number of young vines and replacements as compared with vineyard No. 1, the reduction of yield on the whole area is much less than the average reduction in yield on the vines attacked.

The studies described above show quite clearly that there is a marked difference in the degree of infection found in different varieties grown in the Niagara peninsula. This difference is in all probability due to varying degrees of resistance to the disease in the varieties themselves. Some of the most important commercial varieties are most severely attacked; our most important white variety, Niagara, appears to be highly resistant. Young vines are rarely attacked and typical leaf and arm symptoms do not often occur on vines under ten years of age.

The effect of the disease upon the yield does not seem to be appreciable until leaf symptoms have developed. After that there appears to be a decided reduction in crop. The most serious effect of the disease is the death of the vines, leading to a reduction in yield which is not wiped out until the replacements come into full bearing. The difficulty frequently experienced in bringing along replacements in a full grown vineyard adds to the serious effect of the disease.

Symptoms of the Disease

As Reddick (2) has already described rather fully the symptoms of Dead Arm, I shall deal with the subject but briefly. The most easily recognized symptoms are (a) the appearance of dead arms on vines and (b) the appearance of vines dead, at times practically to the ground, with suckers growing up from the base. Plate III, Figure B shows a Moore's Early vine with the two upper arms killed by the disease. The two symptoms mentioned usually become evident in the spring when the vines begin to put out their new leaves. Thus the death commonly occurs in the winter. The death of vines or arms in the summer seems to be a very rare occurrence indeed. Once the leaves have come out, growth almost invariably persists throughout the summer. Of the large number of attacked vines examined during 1926 and 1927 only one case has been observed where the leaves on an attacked arm actually dried up and died. This vine was noticed at the end of September, 1927, after a prolonged period of drought.

Next to the above, the most readily recognizable symptoms are those of the leaves and branches. The examination of an attacked vine in June or early July shows the young branches or shoots from the affected arm stunted, with shortened internodes and an erect habit. This is shown very clearly on Plate I and on Plate III, Figure A, which reproduce photographs of an Agawam and a Concord vine respectively. The leaf symptoms are equally striking. They are small, misshapen and chlorotic, as is illustrated in Plate IV, Figures A and B. The former figure shows an affected shoot from a Campbell Early vine with a healthy shoot for comparison. As will be seen.

the diseased leaves are much less than half the size of the healthy ones. They show marked chlorosis except along the veins. They are abnormal in shape and are curled and crinkled. Figure B. shows diseased leaves from a Concord vine. Here another feature is evident in the death of leaf tissues between the veins, which frequently leads to a shredding of the leaves as illustrated at the right side of the figure.

Figure A. also shows the general stunting effect of the disease. Comparative measurements of the internodes of diseased and healthy arms (bearing canes) and the shoots borne by them, have given quite interesting results.

Measurements were made on vines of the following varieties: Concord (3), Campbell Early (1), Agawam (2) and Worden (1), the number in brackets indicating the number of separate measurements made on each variety. All the internodes on a bearing cane and all the internodes but the last two on the shoot were measured, these last two being ignored because they were still elongating. Measurements were made on July 9 and July 17, and are recorded in Tables 8 and 9, averages only being given.

TABLE 8.—*Average length of internodes on bearing canes in diseased and healthy condition.*

Variety	Healthy Cane	Diseased Cane	Ration of diseased to healthy
Concord	3.8 in.	3.03 in.	.797
Campbell Early	2.45 in.	2.44 in.	.996
Agawam	3.79 in.	2.6 in.	.686
Worden	3.73 in.	3.10 in.	.830

TABLE 9.—*Average length of internodes on diseased and healthy shoots.*

Variety	Healthy Branch	Diseased Branch	Ratio of diseased to healthy
Concord	4.05	1.72	.425
Campbell Early	1.91	1.70	.890
Agawam	3.65	1.55	.425
Worden	2.70	1.97	.729

These tables show the progressive effect of the disease in stunting growth. The cane of the previous year which had been kept for bearing shows a comparatively small but distinct stunting as a result of disease in three of the four varieties tested. The growth of the present year (1927) reveals a much greater stunting, as is shown in the last column of Table 9.

The effect of the disease on the diameter of the branches is not so marked in the early stages of growth. However, the difference becomes very pronounced before the end of the growing season. An anatomical study of diseased and healthy branches shows the reason for this as well as revealing a number of other interesting features connected with the disease.

Plate V, Figures A and B, reproduces microphotographs of cross sections taken near the base of a healthy and a diseased branch respectively, the variety studied in this case being Concord. The magnification is the same in each case so these figures are in every way comparable. Nevertheless, these sections would hardly be recognized as belonging to the same species. Figure A shows the normal development of a branch at the end of June. The wood is well developed with both vessels and fibres showing lignification. The medullary rays are narrow. The cambium, somewhat torn in the section, indicates active division. No fibres have as yet been differentiated in the phloem but immediately external to each phloem bundle, lies a bundle of pericyclic fibres which are already strongly lignified. The cortex is not strongly developed.

If we turn to the section of a diseased twig we find conditions very different. In the first place both wood and phloem are very much less developed than in the healthy branch. In the wood there is a very meagre development of fibres and practically no lignification is present except in the walls of the vessels. The medullary rays are broad and consist of very thin-walled cells. The cambium is narrow with little evidence of activity, and while the pericyclic bundles are present they are unlignified. Finally, the cortex is much thicker than in the healthy branch. The whole structure of the branch suggests the character of an herbaceous rather than of a woody shoot.

Sections of similar branches made on August 22, 1927, revealed much more striking differences. The differentiation of tissue in the diseased branch remained practically at the stage illustrated in Plate V, Figure B, and the diameter had not appreciably increased. The cambial layers showed practically no indication of activity and there had been no noticeable change in pericyclic bundles, phloem or wood. In the healthy branch, on the other hand, the amount of secondary wood had greatly increased and a great differentiation had taken place in the phloem, as many as three rows of fibres (hard bast) having appeared. The cell walls of the pericyclic bundles had increased in thickness and the cambial region indicated still pronounced activity. Associated with these structural differences was a very much greater accumulation of starch in the parenchyma of the diseased than in that of the healthy branch, indicating, on the part of the former, an inability to utilize properly the products of photosynthetic activity.

Reddick states that leaf chlorosis is present only in June and early July, while the other leaf symptoms persist. This is hardly correct. What actually happens is this: Chlorotic symptoms are usually confined to the primary leaves of a diseased branch. In July, secondary branches develop from axillary buds and these in turn bear leaves which are almost invariably normal in colour or at most a slightly paler green. They are commonly somewhat stunted and they may be curled, though this is by no means always the case. An examination made in late July or August shows that many of the primary leaves have dropped off. Those which persist retain their chlorotic character and commonly show even more necrosis and shredding than they did in June. In our experience they never recover. It is the development of secondary leaves and the dropping of many of the primary ones which mask the early symptoms and lead the grape grower to believe his vine has recovered. Plate II, reproducing a photograph taken towards the end of August, shows the later leaf and branch development of the Agawam vine figured on Plate I. The leaves on the upper arms which are visible in the picture are normal in colour but are dwarfed, as a comparison with those on the lower arms shows clearly. Plate VI shows a portion of a diseased branch bearing two primary leaves which are distinctly chlorotic.

The upper leaf carries in its axil a developing secondary branch which bears leaves of a normal colour. The material photographed is from a Moore's Early vine, one of our earliest varieties. This accounts for the development of secondary leaves so early as July 11 when the photograph was taken.

Other symptoms mentioned by Reddick are (a) "peculiar longitudinal ribbed excrescences on the trunk or arm" and (b) "small reddish brown or black spots on the green shoots, petioles, peduncles and leaf veins" which he states are very characteristic. As to the former I have not been able to find the excrescences mentioned. With regard to the latter, while such spots are no doubt to be found, I have seen them but rarely and under microscopic examination they have not revealed the presence of any fungus mycelium. Under the conditions existing in the Niagara peninsula I regard this as an unimportant and unreliable symptom.

Finally, we come to what must be looked upon as the most important symptom of all. This is furnished by the necrotic lesions or cankers which are to be found almost invariably on trunk or arms of a vine attacked by the disease. These lesions seem to have been largely overlooked by Reddick, though, in his earlier bulletin (pp. 339 and 340), he describes necrotic lesions on the stem. He refers in both his papers to a dry rot in the heart of the trunk and usually extending to the margin as more or less characteristic. The stem necrosis is, however, not primarily one of the heart nor does it show the character of a dry rot except in advanced stages when secondary organisms have gained an entrance. Plate VII, Figure D, shows a typical stem lesion which here surrounds the stub of a branch removed some time previously. These lesions, whether they are on stem or arm, are, in my experience, invariably associated with the stub of some branch or can be traced to some pruning wound. They show, as in the figure, a zonation due to the successive annual attempts of the vine to overgrow the necrotic area. Plate VII, Figure C, shows a cross section of a very advanced stem lesion in which practically the whole of the stem has become involved, a very small portion on the upper side being still alive. The section shows a striking zonation marking the annual progress of the disease. Secondary rot has progressed some distance on the underside of the section while it has begun above from a crack in the stem. These lesions are frequently very extensive, reaching up and down the stem for a distance of three feet or more and at times, as in the case illustrated, embracing all but a minute portion of the stem. Not infrequently two lesions occur on opposite sides of the stem, meet in the centre and so leave two areas of healthy wood on the sides lying at right angles to them. On Plate I a lesion can be seen on the right stem branch extending from about a half inch below the lower wire along the right side up to the top where the two upper arms take off. Similarly, on Plate III, Figure B, a stem lesion extends from the point where the knife is stuck into the stem, right up to the top. As already noted these stem and arm lesions are to be found on vines which show no other symptoms. In fact they may be quite extensive and have existed for at least three years before any leaf symptoms occur. They may be quite far removed from the arms showing symptoms and are undoubtedly, in the vast majority of cases, the real seat of the disease.

The lesions already described are those accompanying fairly advanced stages of the disease. In early stages the lesion in section quite frequently appears as a wedge-shaped darkened area coming to a point in the centre of the stem. This is illustrated in Plate X, Figure C, where the section has been taken some little distance from the centre of the lesion. As will be seen, the margins of the discoloured area follow rather closely the medullary rays and there is quite an abrupt transition from the diseased to the healthy tissue. There is in this stage no sign of dry rot and in fact, as will be shown later, the causal organism appears to exert no appreciable action on the cell walls of the wood. Any definite breaking down of tissue must be the result of the action of secondary organisms.

The stem lesions are, in most cases at least, associated with pruning wounds of considerable size and my observations lead me to believe that infection rarely takes place through a wound produced by the removal of a one or two year old branch. This would help to explain the comparative immunity of young vines, for it is only after a vine has reached five or six years that any considerable number of large pruning wounds will be made. It is interesting to note in this connection that Brooks (9), in his work on Silver Leaf disease, found that wounds in three or four year old branches were more frequently infected than those in one and two year old shoots.

This important symptom is naturally one that can be identified only after the removal of the old dead bark, which accounts for the fact that it is overlooked by grape growers. An examination of the stems and arms of infected vines, after this has been done, shows the presence of lesions in almost every case. In the examination of several hundred diseased vines we have failed to find a stem or arm lesion in only one case. There the infection had taken place on the cane of the previous year which had been kept for bearing and was confined to it.

Cane infection, which Reddick apparently looks upon as the chief source of spread and which he illustrates clearly in his bulletins is, I believe, a very rare occurrence in the Niagara peninsula. It is possible that under certain climatic conditions this may prove to be a more important phase of the disease than our observations indicate, but of the two seasons over which these observations have extended, that of 1926 was a decidedly wet one such as one would expect to favour this type of infection. I am inclined, therefore, to look upon cane infection as a very minor means of spreading Dead Arm in our grape growing area. This view is strongly supported by the fact that the disease rarely occurs on young vines. Were cane or twig infection important there seems no reason why the disease should not be as prevalent on young as on old vines.

Gregory (7) reports the disease as occurring on the berries and both he and Reddick have been able to produce the disease on berries by artificial inoculation. We have not found the disease on berries in the field but our observations on this phase of the disease have not been extensive. The fact that according to Reddick the disease on berries is difficult to distinguish from Black Rot makes berry symptoms of little value to the grape grower.

The Cause of the Disease

As already shown by Shear (*5*) and Reddick (*1, 2*), the cause of Dead Arm is the fungus *Cryptosporella viticola* Shear. After the descriptions given by these two authors it is unnecessary to deal more than briefly with the subject here. I shall, therefore, confine myself mainly to points where my observations may differ from or supplement those of the authors mentioned.

A fairly extended search in the spring of 1927 failed to disclose the perfect stage of the fungus. The imperfect fruiting stage (*Fusicoccum viticolum*) is present in abundance on most stem and arm lesions, the pycnidia being usually arranged in rows more or less parallel to the long axis of the organ upon which they are found. Plate VII, Figure A, showing pycnidia breaking through the bark on an arm of an attacked Niagara vine, illustrates this. Reddick states that the pycnidia on woody parts are to be found most abundantly in the early spring and that later, after the June rains, only the bases of pycnidia remain. This is not entirely in accordance with observations here. Pycnidia can be found from May till September and throughout this period, after one or two days of high humidity, some of them can, almost invariably, be found emitting spores, often in long spiral masses as illustrated in Plate VII, Fig. B. The first spore emission observed in 1927 was on May 22 but as the spring of this year was a backward one and as the observations were made on the Horticultural Experiment Station, where the season is usually a week or ten days later than farther from the lake, spore emission probably begins normally about the first of May in our earlier grape areas. The spores appear as a yellowish white mass at the apex of the pycnidium. This may be quite simple in shape or may take the form of a long corkscrew-like body, as illustrated in the figure. As noted by Reddick, the masses harden to a horny consistency under dry conditions but readily soften under moisture and dissolve in a drop of water. Plate IX, Figure B, shows a section of one of the simpler pycnidia as it appears under a magnification of about 75 diameters. Spores are being emitted but the emitted mass has, for the most part, been washed away in making the preparation. Plate IX, Figure A, shows in section another type with a long extension under the bark. Multilocular or convoluted pycnidia such as those described and figured by Reddick, although they occur, do not seem in our material to form the preponderating number. Plate IX, Figure C, reproduces the centre of a flask-shaped pycnidium much more highly enlarged (about 370 diameters). This figure shows the mode of formation and attachment of conidiospores, as also their mode of exit from the ostiole of the pycnidium. Only the elliptical conidiospores can be made out in this figure, and as a matter of fact in preparations made during 1927, the long scolecospores or pseudo-paraphyses described by both Reddick and Shear as characteristic of this form have been inconspicuous both in collected and in pure culture material. However, they were to be met with here and there. In 1926 they were found quite abundantly so it would seem that they vary in number under different conditions.

Plate IX, Figure A, shows, in addition to a pycnidium, another structure which, so far as I am aware, has not been previously described. To the left of the figure is to be seen a mass of hyphae which, like the pycnidium, has burst through the cork but which has remained quite sterile. Structures like these are not uncommonly to be found scattered among the pycnidia. They are probably to be considered as aborted pycnidia, though they may, conceivably represent rudimentary perithecia.

Reddick's conception, which he has developed more especially in his second bulletin, of the causal organism as one mainly attacking young growing tissue, has, it seems to me, led him to neglect the study of the fungus as it occurs in the older woody parts. The fact that I have been forced almost from the start to look upon the organism as primarily a wound parasite has led me to pay particular attention to this.

An examination of sections (radial sections are the most instructive) through a stem lesion, more especially where it approaches healthy tissue, shows features which are illustrated on Plate VIII. In the wood, the fungus is most abundant in the parenchyma including the medullary rays. It is by no means absent from the vessels as is shown in Plate VIII, Figure E, but it cannot be looked upon as a vascular parasite, its presence in vessels being apparently more or less accidental. In the wood fibres also hyphae can be found here and there, but these elements are for the most part free. On the other hand in the wood parenchyma and medullary ray cells the growth is luxuriant. A striking feature of the attack on these cells is the disappearance of starch grains from cells actually penetrated by the fungus where sections are taken in the autumn or early winter from near the margin of a lesion. At this time the medullary ray cells and wood parenchyma of healthy tissue are loaded with starch. At the margin of a lesion one finds the starch absent from the cells actually penetrated as well as from neighbouring cells on all sides. This indicates either that starch actually formed in these cells has been dissolved by the fungus or that the translocated carbohydrates have been drawn upon by the fungus before the actual formation of starch. That many of the parenchyma cells in this region are still capable of storing starch is shown by the presence of scattered groups of cells loaded with it. The fungus is able to dissolve starch as is indicated by the reaction of a starch-containing medium such as potato dextrose agar on which it is growing. Plate X, Figure A, shows a plate culture on this medium which has been flooded with iodine. The solution of starch extends out some distance beyond the fungus growth.

Associated with the absence of starch, one finds in these parenchymatous cells quite large amounts of a yellowish brown substance extremely resistant to solvents and chemicals in general, which has the characters of what is known as wound gum. This may occur as granules (Plate VIII, Figures A and B) or as larger more or less spherical masses (Plate VIII, Figure C). It is the formation of this substance, together with a darkening of the cell walls, which gives the brown colour to the tissue in the lesions. Wound gum is not confined to the wood parenchuma. It is found also in the vessels, especially where these are not plugged by tyloses which are formed abundantly in invaded tissue. Plate VIII, Figure D, shows a longitudinal section of two vessels of which the right one has its lower portion plugged with wound gum. Both vessels have been invaded by fungus mycelium which can be seen actually embedded in the wound gum. This substance is also found in the phloem where it is confined to the sieve tubes and the phloem parenchyma.

The question of wound gum formation in the grape and its relation to tissue invasion by the fungus is at present under investigation. The recent work by Swarbrick (*8*) has drawn renewed attention to the question of wound gum formation and it seems that we have here to do with a protective process which may have an important bearing on the invasion of wounds by wound parasites. Brooks' (*9*) work on Silver Leaf Disease appears to point in that direction and my preliminary observations on the invasion of wounds by *Cryptosporella viticola* lead me to suspect that wound gum may, under certain circumstances, perform a protective rôle in the grape vine. The fact that fungus hyphae can be found embedded in wound gum as illustrated in Plate VIII, Figure D, would seem to be against any such theory, but it is quite possible that the gum has been deposited in the vessel after invasion by the fungus.

In the invaded tissues the fungus seems always to be intracellular. The penetration of cell walls is invariably through the pits, the thicker hyphae, quite commonly found in medullary rays and wood parenchyma, being greatly constricted where they pass through the wall (see Plate VIII, Figures A & B. The microscopic picture carries no suggestion of a solution of the cell wall but rather of the mechanical piercing of the middle lamella at the pits.

When we turn to the phloem, conditions seem to be somewhat different. In the soft bast and medullary rays of the phloem the fungus appears to grow more luxuriantly and spread more rapidly than in the wood. As a matter of fact a surface examination at the end of an advancing lesion frequently shows the phloem discoloured a dark brown, while the wood beneath is almost normal in appearance. Both sieve tubes and phloem perenchyma are penetrated and this soft tissue is early so broken down that it is difficult to get satisfactory sections for examination. Plate X, Figure B, shows a radial section through a portion of the phloem region of a lesion. The dark portion to the right is the cambial region greatly discoloured and collapsed. Then comes a zone of soft phloem with a sieve tube invaded by hyphae. Left of this is a zone of phloem fibres which, apparently, are never invaded by the fungus. Next to this is another zone of soft phloem. Here a sieve plate with its callus plug is shown, the section having been made in the winter, and a fungus thread is embedded in the callus.

It has not been possible to decide as to whether the piercing of the cellulose walls is a purely mechanical process or whether solution of the cell walls also takes place. Observations indicate that mechanical pressure plays a rôle but there is here no evidence of hyphal constriction at the point of penetration. The action of the fungus on cellulose and other compounds which go to make up the cell walls of the grape vine is at present under investigation.

As the fungus seems unable to penetrate the hard fibres which, in the grape, form concentric rings in the phloem broken only by the medullary rays, it must make its way outward by these latter. It forms dense masses under the youngest cork layer from which the pycnidia as well as the sterile bodies mentioned above develop. These gain access to the outside by a bursting of the young cork layers, as has been described by Reddick.

As has already been stated, the stem lesions may be situated at a considerable distance (at times two or three feet) from the arms or branches which show Dead Arm symptoms. These in the great majority of cases show not the slightest evidence of fungus invasion and are in section quite normal in colour. In every case, however, the lesion is to be found in the line of conduction to the affected arm. The leaf and branch symptoms already described show clearly that the metabolism of that part of the vine has been severely disturbed, but whether that has been due to the production of a toxin by the fungus or to the reduction of reserve food material, or to both, is not clear. Injection experiments with extracts from diseased tissue made in 1927 were not successful, and it is planned to repeat these in the spring of 1928. The fact that a large stem lesion, such as is commonly to be found in association with marked leaf and branch symptoms, must represent a heavy reduction in the amount of reserve material upon which the young buds and branches have to depend for growth, and the further fact that the severest symptoms are generally confined to the primary leaves, while the secondary ones are much more nearly normal, leads me to believe that the absence of reserve materials is an important factor in producing the symptoms described. It must not be forgotten, however, that although the later leaves may develop almost normally, the branch tissues remain in an undeveloped state which shows clearly that, whatever may be the cause of the disturbance, it continues to act throughout the growing season.

Pure Culture of the Fungus

The isolation of the causal organism presents no difficulties. At almost any time during the summer pycnidia containing fully developed spores can be found on attacked vines and material placed in a moist chamber for twenty-four hours will commonly yield masses of emitted spores. A transfer from such a mass with a pouring of plates will give abundance of uncontaminated colonies in the course of two or three days, at ordinary room temperatures in the summer.

A study of the nutrition of the fungus has been undertaken and will be reported upon later. It may be noted here that the most satisfactory media used up to the present are oat agar and potato dextrose agar. Prune agar and malt agar are less satisfactory while the growth on ordinary nutrient agar is meagre. Roll cultures in ordinary Erlenmeyer flasks using oat agar as a medium have been found particularly suitable for obtaining large quantities of spore material for inoculation purposes. Pycnidia develop very freely and, under ordinary summer temperatures in the laboratory, spore material is available in the course of from four to six weeks.

Inoculation Experiments

Gregory (*7*) in 1913, obtained infection by spraying spore suspensions on to green shoots and berries. Reddick (*2*), after an earlier failure, was able to confirm these results. He obtained fairly convincing results by inoculating with a saw which had been previously drawn a few times through a stem lesion. He also produced infection from mycelial inoculation into cuts made in stems and bearing shoots, but failed in his attempts to inoculate roots. As the results of field study pointed clearly to inoculation through pruning wounds as the ordinary method in nature, my inoculation experiments have been largely confined to this. In these experiments either a fresh wound was made by cutting off a branch or the surface of a wound made during the pruning season was cleaned off by scraping. The fresh wounds were either inoculated at once or left for a couple of weeks to dry. A spore suspension in sterile distilled water, usually from a pure culture, was then sprayed on to the wounds with an atomiser. In the earliest experiment the inoculum was painted on with a brush, an atomiser not being available at the time. The first inoculations were made on July 22, 1926, in the vineyard of the Horticultural Experiment Station at Vineland. A row containing the following varieties was used for the purpose:—Concord (2), Cottage (2), Niagara (2), Vergennes (2), Worden (2), Campbell Early (2), Lindley (3). One of the Campbell Early vines was so badly diseased as to be unsuitable and was discarded.

On each vine two branches were cut back; one cut end on each vine was inoculated immediately, the other being left for two weeks. As stated already in the first cases a small brush was used for distributing the spore suspension; in the second case an atomiser was used. The inoculated ends were covered with moistened absorbent cotton which in turn was protected from drying out by a covering of waxed cloth.

On July 26 a similar set of inoculation experiments was started in a vineyard of Concord grapes belonging to the Ontario Grape Growing and Wine Manufacturing Company, Ltd., St. Catharines. Here twelve inoculations were made on six vines in the manner described above. The first observations of those inoculations made on August 10 were to verify the presence of mycelium in the inoculated stubs. In all cases where sections were taken from these mycelium having all the characteristics of *C. viticola* was found, the penetration varying from one-quarter to three-quarters of an inch.

Observations were carried out at intervals of from two to three weeks up to October 6, 1926, and at the Vineland vineyard on January 15, 1927. There were distinct signs of progress of the disease up to October 6, but very little after. In a few cases typical pycnidia developed on the infected stubs. These were found as early as September 23 or almost exactly two months after the inoculation.

An examination of the Vineland inoculations on May 4, 1927, (those at St. Catharines had unfortunately been removed during the winter pruning) showed 13 out of the 28 inoculated stubs bearing pycnidia. The first emission of conidiospores from these was observed on May 23 and from this material pure cultures were obtained which yielded spore material for further inoculations. Other work occupied the summer so the next careful examination was delayed till September 2, 1927. At this time 5 inoculations out of the 28 originally made had led to a development of the disease beyond the bases of the original inoculated stubs for distances varying from 1 inch to

20 inches. It can, I think, be safely concluded that we have here the beginnings of the necrotic lesions typical of the disease. In the case of the other inoculations made it seems doubtful if these will now lead to infection of the vines. It looks as if the vines in these cases had been able to throw a barrier across at or near the base of the stub inoculated. While time has not allowed for a careful microscopic examination of these cases I think it probable that the formation of wound gum has had much to do with the stopping of the parasite's progress. This may possibly act in several ways. In the first place the disappearance of starch from the areas where wound gum has formed indicates, as many authors (Swarbrick and others) have suggested, that the latter is formed at the expense of the former. It seems highly improbable that the fungus is able to utilize the wound gum itself as food material so its formation may quite conceivably lead to the starvation of the fungus or at least to the severe checking of its growth. In the second place, the formation of wound gum and tyloses block the vessels very effectively preventing the exit of dissolved materials from the stem. Swarbrick's experiments on apple branches in which he attempted to suck coloured solutions through them by means of a suction pump were repeated by us on grape stubs. Stubs which had been left exposed for a month in the summer were found thoroughly blocked. Finally, it is possible that wound gum and tyloses form something of a mechanical barrier to the progress of the parasite. Preliminary microscopic studies indicated, however, that such a mechanical barrier, if at all existent, is an imperfect one. As already shown in Plate VIII, Figure D, fungus hyphae have been found actually embedded in the wound gum of vessels while the arrangement of the wound gum masses in the medullary ray and wood parenchyma cells is not such as to suggest a mechanical barrier. In fact here also hyphae have been found in individual medullary ray cells along with wound gum. As to the possible protective action of tyloses, here again hyphae have been found penetrating their walls and passing between them in the vessels. The whole question of the relation of wound gum formation to the progress of the parasite is an extremely interesting and important one and, as has already been stated, is forming the subject of special study.

In order to study early stages of penetration, inoculations on freshly cut stubs have been made during the summer of 1927, and the stubs have been removed at intervals of from 2 days to 2 weeks. To prevent as far as possible the intrusion of other organisms the surface of the branches was cleaned with a solution of 95 per cent alcohol before the cut was made, the spores were sprayed on in a suspension of sterile, distilled water and the wound was covered immediately with absorbent cotton, moistened with sterile, distilled water, and then with waxed cloth.

The microscopic examination of the inoculated stubs is as yet incomplete. However, sections of stubs made four days after inoculation have shown germination of conidiospores in the vessels with the formation of germ tubes of considerable length. Plate VIII, Figure E, shows two germ tubes of *Cryptosporella* conidiospores in a vessel of one of these stubs. Plate X, Figure D, shows under higher magnification a spore germinating in a vessel. These germinating spores were found at a distance of about ½ mm. from the cut end and were no doubt drawn in by capillary action. Had the cut surface remained exposed the spores would probably have been drawn in still farther. These results are very similar to those already recorded by Brooks and Moore (*10*).

A large number of further inoculations have been made during the summer of 1927, the results of which will be available only later.

CONTROL MEASURES

Control measures should be considered under two headings: (a) the prevention of fresh infections on vines not already attacked, and (b) the removal of diseased tissue from vines suffering from the disease.

As already stated, by far the most important mode of infection seems to be through pruning wounds. The fact that young vines rarely show the disease even to the extent of stem lesions indicates that the larger pruning wounds, such as are made on older vines, are the chief channels of invasion for the fungus. Inoculation experiments point in the same direction. Pruning is invariably carried out in our grape areas during the dormant season (normally during January-March). While definite experiments have not as yet been carried out in regard to the possibility of growth of the parasite at this period, it is pretty safe to assume that there is little danger of infection at the time of pruning through the tools used, while the possibility of infection from air-borne spores at this time seems to be excluded. It seems certain that the first natural inoculations synchronize with the bursting of pycnidia and liberation of spores early in May. If, as seems probable, the grape vine is able to protect itself at least partially from invasion by the formation of wound gum and tyloses, it seems likely that, at this time of the year, the process has not proceeded far enough to furnish adequate protection. Swarbrick's work on the apple under English conditions shows that wound gum formation is practically at a standstill during the dormant period and it seems probable that that holds for the grape also. A protection of the wounds for about a month from the beginning of spring growth should tide the vine over the period of greatest danger. Preliminary experiments with various wound dressings, which need not be detailed here, lead me to believe that an early spring spray with Bordeaux mixture would be the most practical means of protecting the wounds. Microscopic examination of tissue under wounds sprayed with Bordeaux or lime sulphur and then inoculated by spraying on spore suspensions showed practically no penetration where the Bordeaux was used, while lime sulphur proved less effective. These experiments must of course be repeated before a definite statement as to the effectiveness of spring spraying can be made. However, I believe spraying with Bordeaux once about the beginning of May, and possibly a second time towards the end of that month, should prove a fairly efficient protection against fresh infection and I would recommend its trial in blocks seriously attacked by the disease.

This is by no means the first time that spraying has been suggested as a control measure for Dead Arm. Reddick suggested that where Black-rot is prevalent the first application for that disease, when the shoots are eight or ten inches long, should protect these shoots from infection with the Dead Arm fungus. This spraying would in all probability be too late to be effective in preventing the infection of pruning wounds. Partridge, (*11*) who recognizes the disease as caused by a wound parasite, states that spraying does not prevent the disease, though the earlier sprays may be helpful in reducing its spread. He, however, does not give any experimental evidence as a basis for his opinion. In the above references the spray indicated is, of course, Bordeaux mixture.

Hiura in the first of his papers cited (*3*) states that the grower in whose vineyard his investigations were carried out used a rather elaborate system of spraying with lime sulphur. The grower started with an application of 1 in 4 or 5 about the middle of April before the buds burst and followed this with three further applications of 1 in 10, 1 in 20 and 1 in 5 or 6, the last being applied to the base of the stem about September (1). Hiura, however, is not prepared to state whether or not the treatment gives full control.

The removal of diseased parts is essential if real control is to be attained. The cutting out of stem lesions, such as may be practised in the case of tree cankers is, of course, out of the question here. All those parts of a vine down to and including the stem cankers must be cut off. If the cankers are situated high up on the stem or, as is often the case where the improved Kniffen system of training is followed, on only one of the stem branches, this will not involve the sacrifice of more than a part of the vine. With the rapidity of growth exhibited by most of our commercial varieties this will lead to only a very temporary reduction in yield. In many cases examined by me, however, the disease had been allowed to develop much too long and the whole vine had to be cut to the base or uprooted altogether.

Unfortunately the removal of diseased portions is associated with a real difficulty of spotting diseased vines. As already pointed out, the disease may be present on the stem of a vine at least two or three years before any definite leaf symptoms appear and the detection of stem lesions necessitates the removal of the old dead bark, a rather tedious process which will not greatly appeal to the grape grower. Stem inspection can, however, be safely restricted to areas around old pruning wounds and I believe a thorough examination of all vines in a badly diseased area is a necessary preliminary step. I would recommend the following procedure: Infected areas should be inspected during June when the leaf symptoms are most readily recognized. This will enable the grower to spot at once the more advanced cases of disease. The stems of all vines should be examined in the regions of old pruning wounds, the old bark being stripped off from these areas. All infected vines should be marked by tying conspicuously coloured strips of cloth below the lower margin of the lesion or of the lowest lesion, where there is more than one. This will enable pruners to spot the vines during the following winter's pruning when all portions above the mark should be removed. Immediate removal may be necessary in some cases, for example where a comparatively small extension of the lesion downwards will involve further bearing portions of the vine. Unfortunately we have no data upon which to judge the probable longitudinal extension of a lesion in a growing season. It no doubt varies with the season and with the variety or even the individual vine. Stem lesions on a number of vines representing different varieties were marked during the early summer of 1927 with the object of getting data on linear growth. Observations will have to be continued for several years before anything like accurate information will be available. As an example of a vine upon which removal could be safely left till the pruning season, the one shown on Plate I may be taken. Here the lower margin of the stem lesion is about a foot and a half from the crotch of the two stem branches so there is little danger of the delay leading to the two lower arms being involved.

Where it becomes advisable to remove diseased portions of vines during June it is necessary to paint the wounds with some protective covering, for the danger of infection is great. As at this time the stems bleed very freely it is somewhat difficult to get an effective covering. Bordeaux paste is of little use as it washes off. White lead proved fairly satisfactory. Probably gas tar would prove more effective but it has not been tried.

These suggestions as to control are only tentative. With the accumulation of more experimental results in subsequent years it should be possible to make the recommendations simpler and more definite. However, I have little doubt that an adherence to the recommendations made above would lead to an effective control of the disease, at no great expense to the grape growers. As far as cutting out measures are concerned, if they are followed consistently for three or four years they will lead, I am sure, to a very marked reduction in the infestation.

CONCLUSIONS

1. Dead Arm disease of the grape caused by *Cryptosporella viticola,* Shear, is the most serious grape disease in Ontario. Estimates made in the field show an infestation on susceptible varieties of between 11 per cent and 50 per cent of the vines in badly attacked vineyards. It is present in all the important grape growing areas of the province.

2. There is a marked difference in susceptibility to this disease among different grape varieties. Unfortunately most of our important commercial varieties are decidedly susceptible. Among these are the two leading early varieties, Moore's Early and Campbell Early and such standard varieties as Concord and Worden. There are a number of varieties which are apparently quite resistant but among these the only important commercial variety is Niagara.

3. While the reduction in yield due to the disease results mainly from the death of the vines and the slowness in growth of replacements in established vineyards, an appreciable reduction seems to occur with the appearance of typical leaf symptoms.

4. The most important symptoms are: (a) death of vines or arms, (b) stunting of growth in leaf-bearing branches and leaves, with the deformation and chlorosis of the latter, and (c) stem and arm lesions associated with old pruning wounds.

5. The view that the chlorotic symptoms which are most conspicuous in June and early July disappear later is erroneous. The primary leaves thus

affected either drop off or are so covered by secondary leaves of practically normal color as to be easily overlooked. Dwarfing and curling are frequently exhibited on these later leaves but chlorosis practically never.

6. The leaf symptoms can be distinguished from ordinary chlorosis by the stunting and deformation of the leaves. Early stages are, however, rather difficult to distinguish, and if the suspected vine shows no stem or arm lesions it should be classed as free from Dead Arm.

7. Associated with the stunting of the branches is a serious change in their internal structure. This consists of a great reduction in all conducting and supporting tissues, with an accompanying absence of lignification. Along with this is a relative increase in the cortical parenchyma and medullary rays.

8. The stem and arm lesions may be present several years before leaf and branch symptoms appear. They are always associated with some pruning wound and bear every evidence of resulting from the action of a typical wound parasite.

9. While the casual organism has been shown by Shear to possess a perfect stage this has not been found in Ontario. The conidial fructifications (pycnidia) are formed abundantly near the margins of the lesions and spores are emitted from about the first of May till the beginning of September, if not later. They must be looked upon as the chief source of infection, the main means of entrance being through pruning wounds.

10. Although the fungus mycelium can be found in all tissues in a lesion excepting the phloem fibres and cork cells, it attacks mainly the wood parenchyma, the medullary ray cells, the cambium and the soft phloem. Its progress is accompanied by a disappearance of reserve starch and an accumulation of wound gum, which latter along with the discolouration of cell walls produces the dark colour of the infected tissue. The fungus apparently does not attack cell walls in the wood and probably not in the phloem. Any appearance of dry rot in a lesion is due to the presence of secondary organisms.

11. Stem lesions may be, and usually are, far removed from the parts of the vine which show leaf and branch symptoms. They are, however, always in the line of conduction to these parts. Leaf and branch symptoms may be due to the production of toxic compounds or to the reduction in food supply to the affected parts or to both. It seems probable, however, that the reduction in food supply to the leaf-bearing branches is the main cause of the symptoms.

12. Inoculation experiments with spores from pure cultures of the fungus on to the stubs of cut branches have led to infection with the production of fully developed pycnidia and the inception of what promise to develop into typical lesions. Attempts at inoculation by spraying spore suspensions on to green twigs, leaves and berries have not been successful, but they have also not been numerous.

13. Control measures suggested are: (a) marking in June the affected portions below the lowest stem lesion and removal at the pruning season, (b) spraying the pruning wounds once or twice in May to prevent infection from spores emitted at that season.

DEPARTMENT OF BOTANY,
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AND
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EXPLANATION OF PLATES

All photographs with the exception of those reproduced on Plate X, Figures A and C, were taken on Eastman's panchromatic films. For macroscopic objects Eastman's K3 filter was almost invariably used. For microphotographs the G filter was employed. The photographs were made, almost exclusively, at the Provincial Horticultural Experiment Station, Vineland, Ontario.

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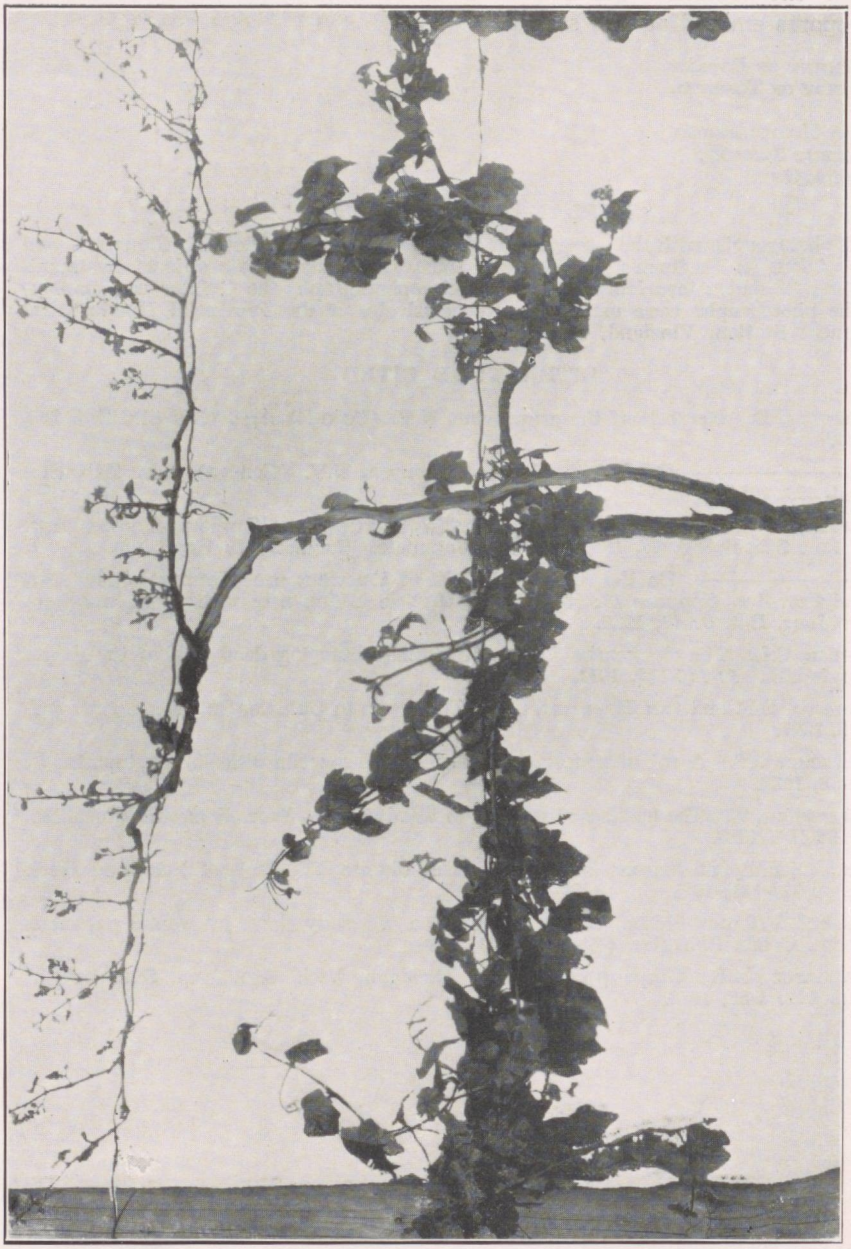

Plate I. Grape vine, Agawam variety, showing typical Dead Arm symptoms on the two upper arms and a long lesion on the stem. Photo 27th June, 1927.

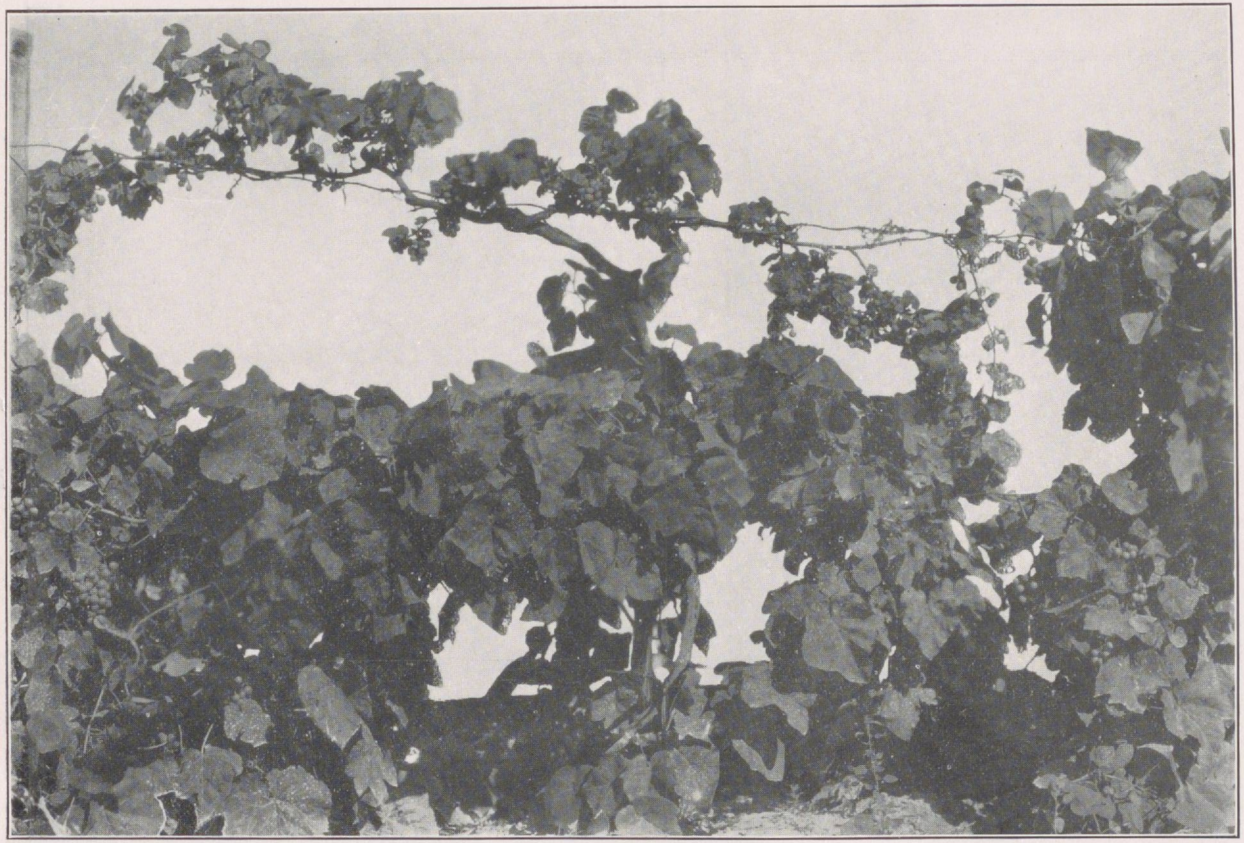

Plate II. The same vine as shown on Plate I. The secondary branches and leaves have developed, masking the symptoms. Photo end of August, 1927.

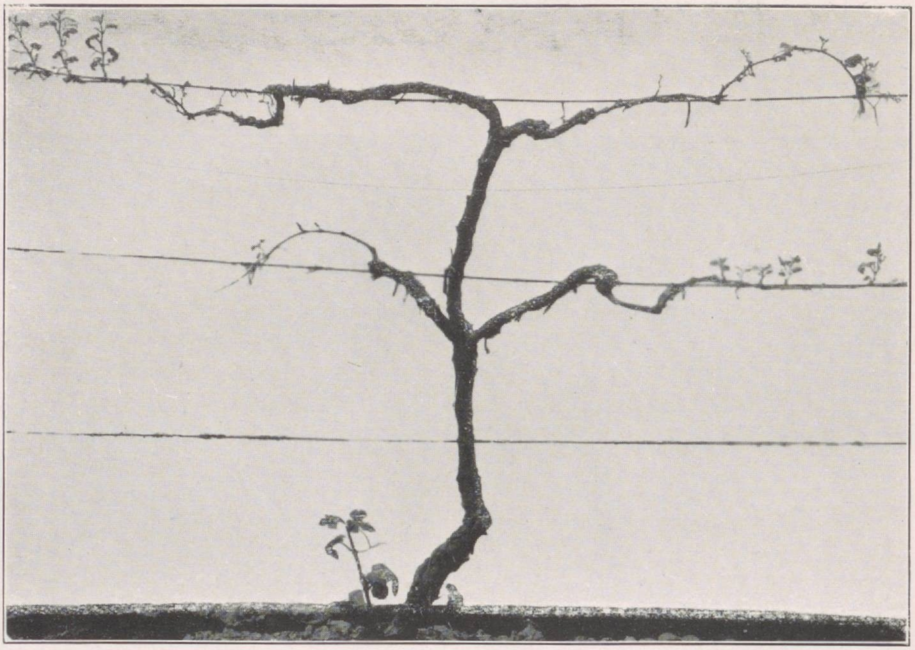

Figure A

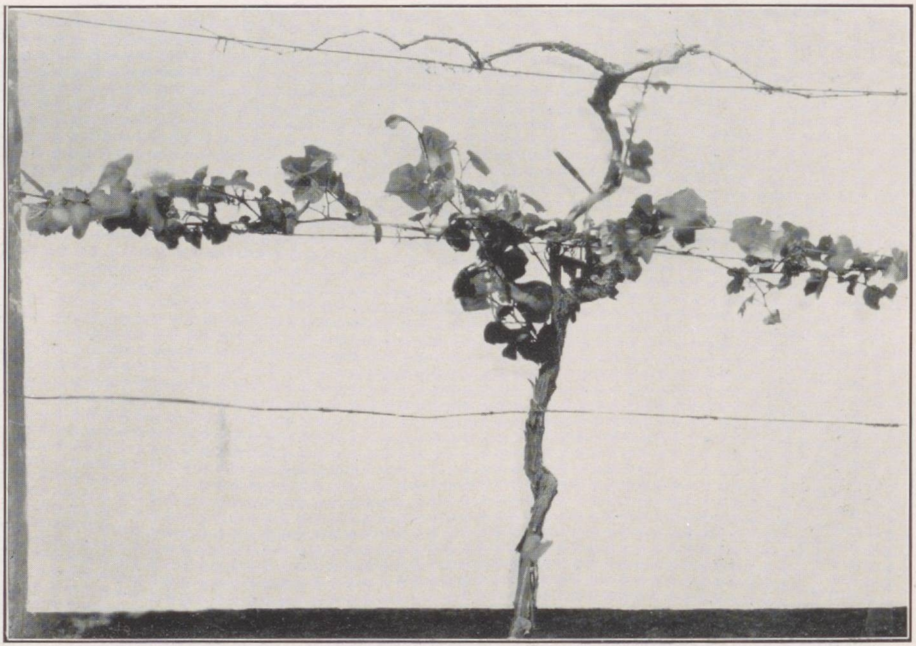

Figure B

Plate III. Figure A. Grape vine, Concord variety, in advanced stage of Dead Arm, showing healthy sucker coming up from base of stem. Photo 24th June, 1927. Figure B. Grape vine, Moore's Early variety showing the two upper arms dead. Lesion extends to top from point where knife is stuck into stem. Photo 5th July, 1927.

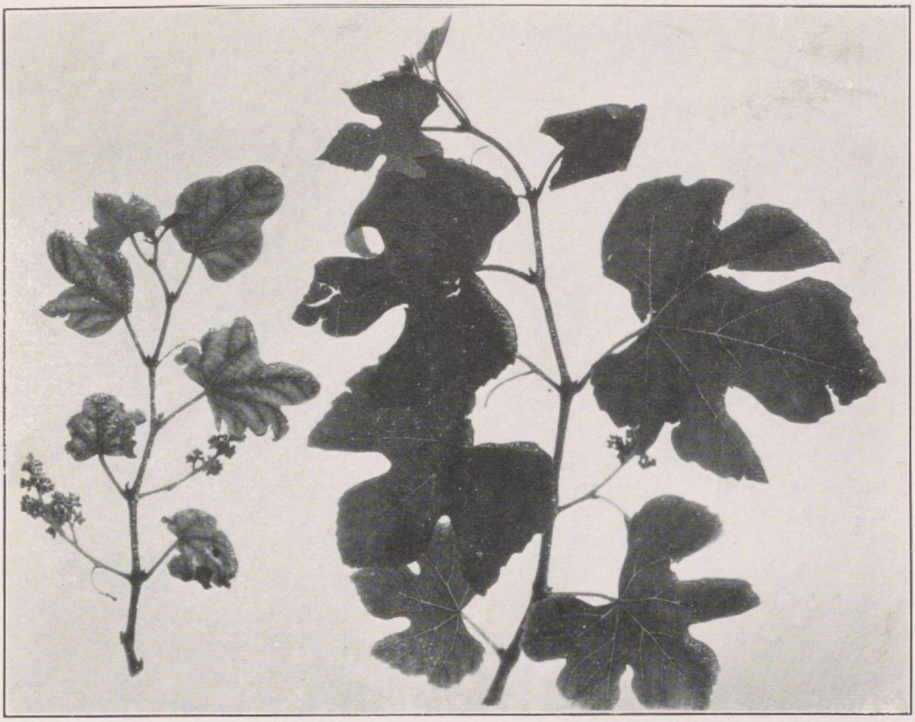

Figure A

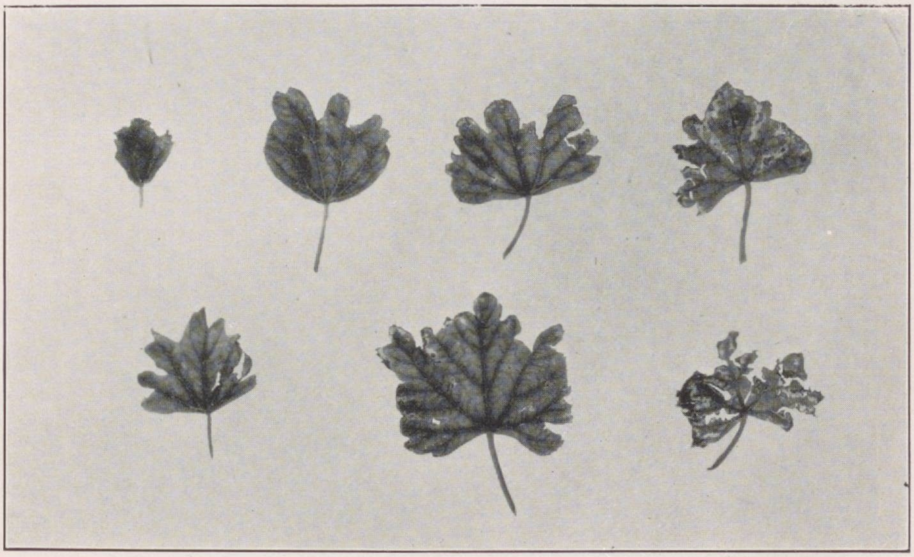

Figure B

Plate IV. Figure A. Diseased and healthy branches of grape vine, Campbell Early variety, to show typical leaf and branch symptoms. Photo 30th June, 1927. Figure B. Leaves from a diseased branch of grape vine, Concord variety, to show chlorosis, curling, malformation, necrosis and shredding typical of disease. Photo 13th July, 1927.

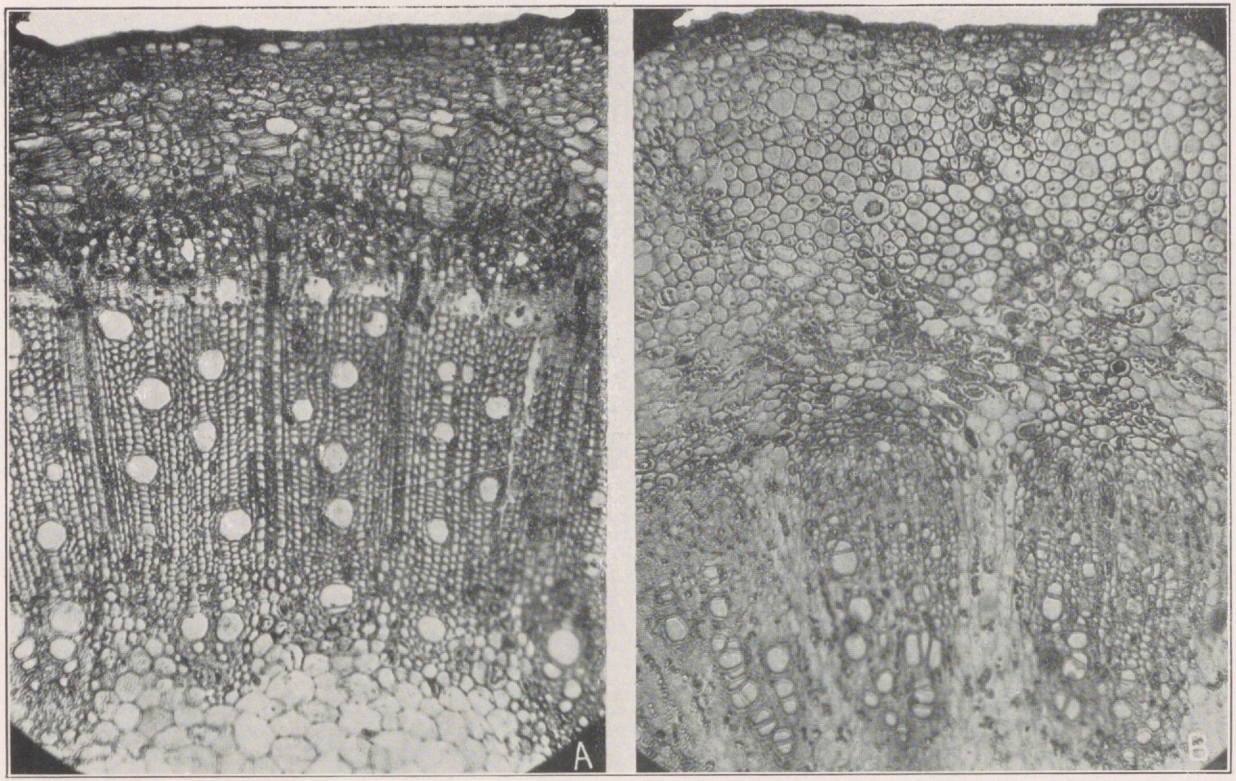

Plate V. Figure A. Cross section of a healthy branch from a grape vine, Concord variety, taken near the base of the branch. Magnification about 25 diameters. Figure B. Cross section of diseased branch from same variety (next vine in row) taken from the same relative position and with the same magnification. Both lots of material collected on 13th July, 1927.

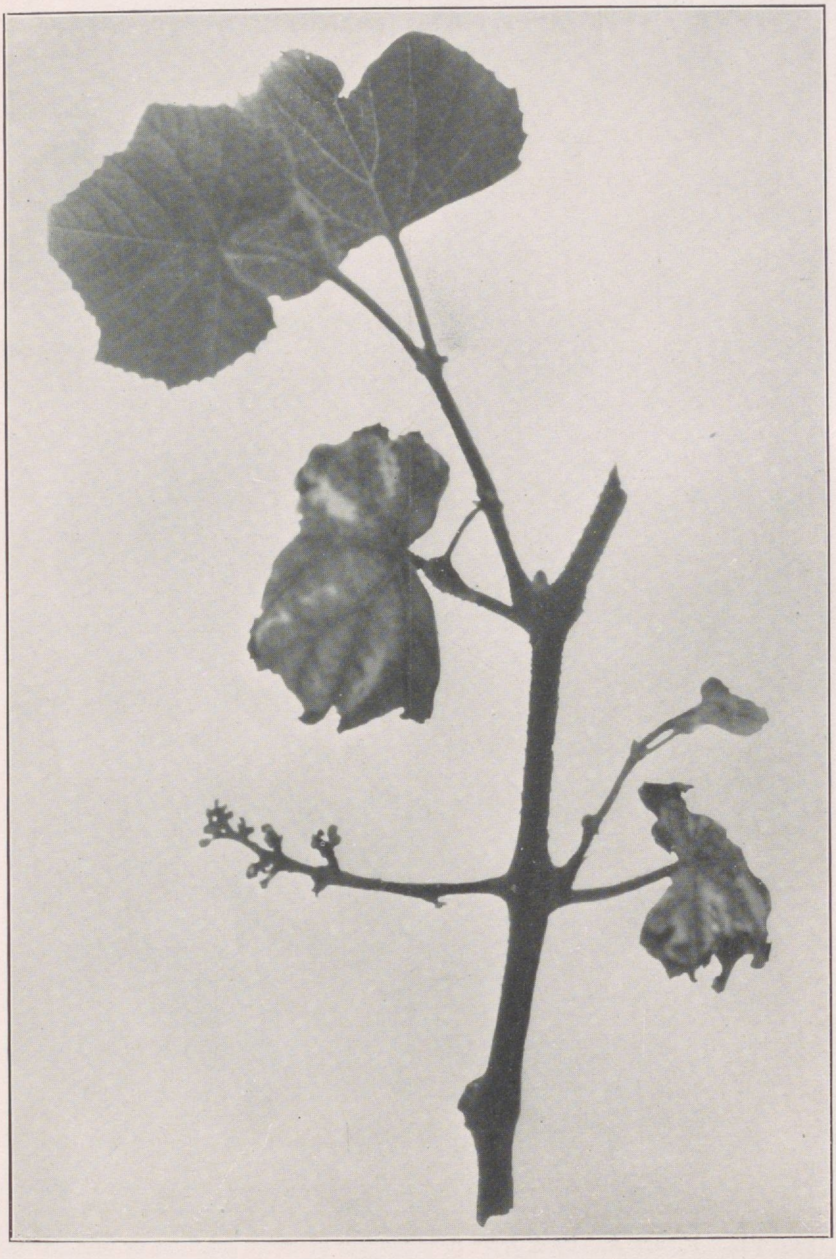

Plate VI. Portion of a diseased branch from a vine, Moore's Early variety. Two primary leaves showing typical symptoms are present. From the axil of the upper one has developed a branch bearing leaves of a normal colour and shape. Photo 11th July, 1927.

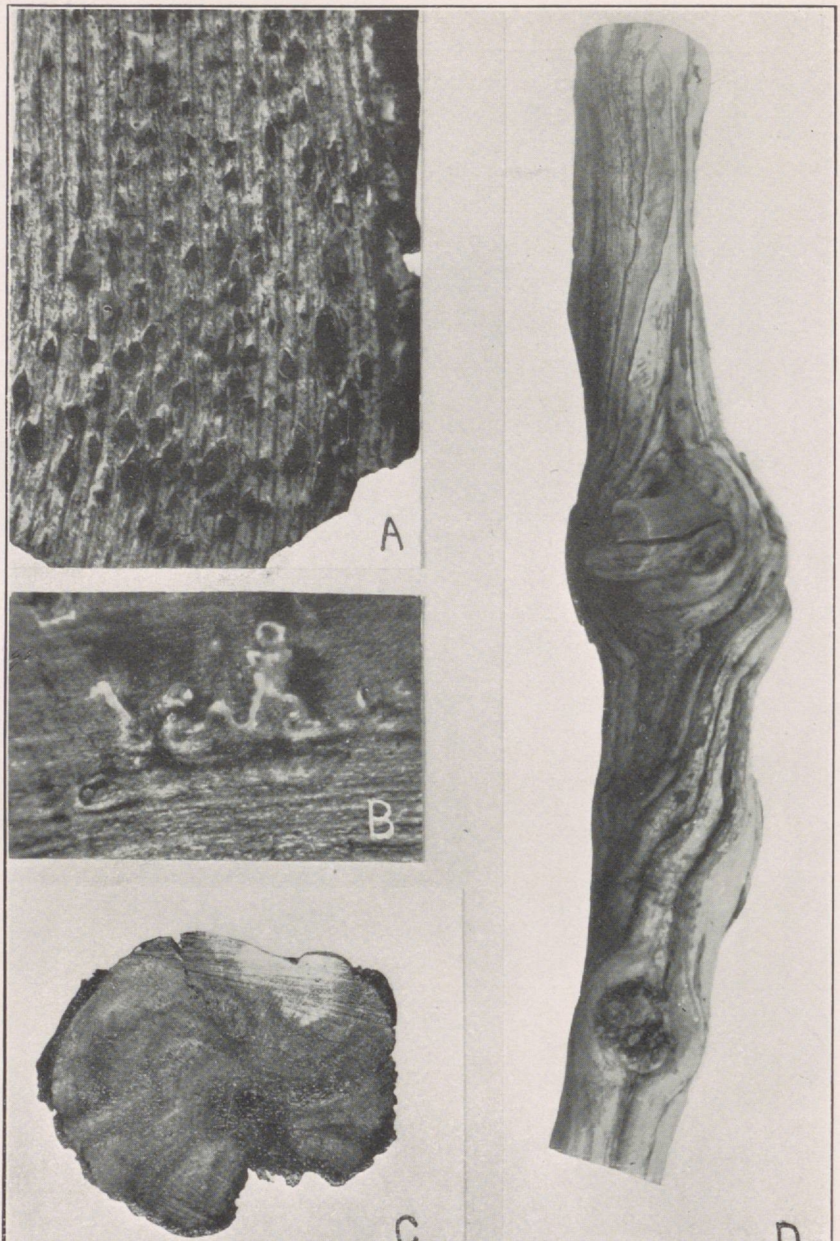

Plate VII. Figure A. Bark from near margin of lesion on a grape arm, Niagara vraiety, to show pycnidia of *Cryptosporella viticola,* Shear, breaking through. Magnification about 5 diameters. Photo 16th July, 1927. Figure B. Small area on bark of stem lesion showing emission of spores in twisted threads. Photo July, 1926. Figure C. Cross section of a grape stem, Concord variety, taken through an old lesion to show annual progress of disease and development of secondary rot. Slightly reduced. Photo February, 1927. Figure D. Piece of grape stem, Concord variety, showing typical lesion about an old stub. Photo February, 1927.

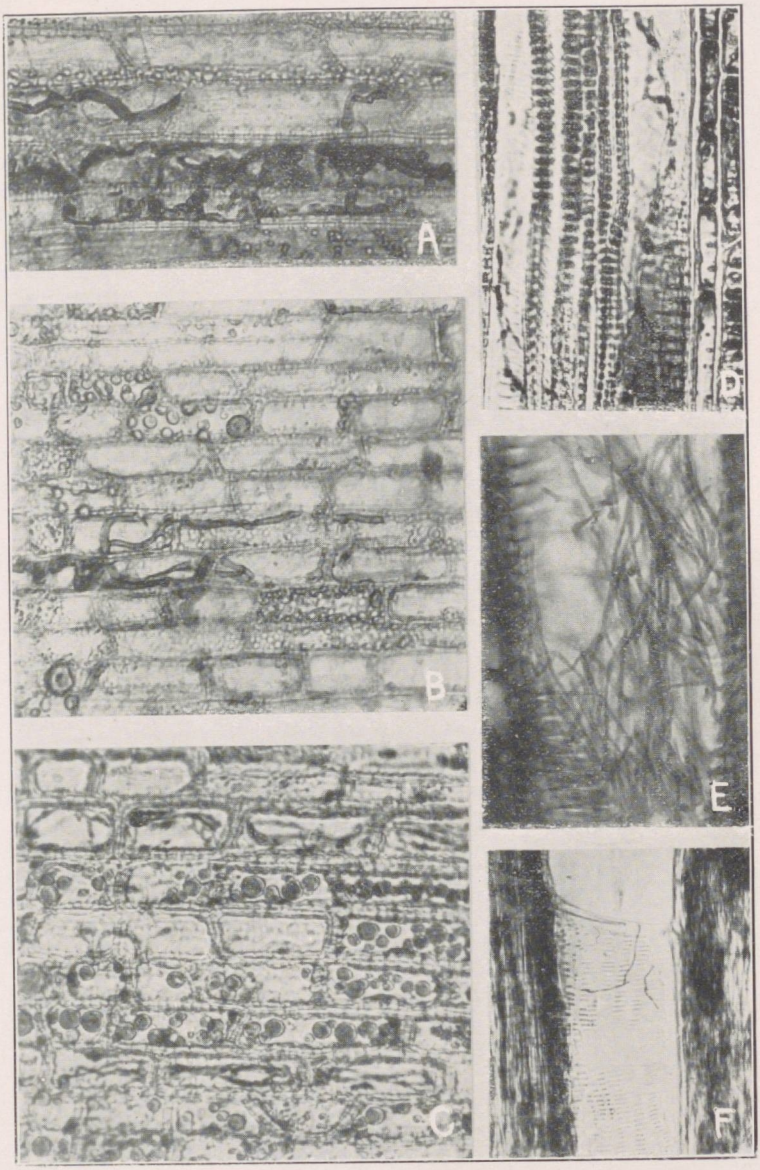

Plate VIII. Figures A and B. Medullary ray cells from near margin of stem lesion showing invasion by mycelium of *C. viticola.* In addition to thicker hyphae passing from cell to cell are finer hyphal threads which show indistinctly. Figure B. shows also wound gum as small granules and larger masses. Material fixed 7th February and stained by Dickson's magdala red and light green method; Figure C. Similar section to above showing invading mycelium and large spherical masses of wound gum. Hand section from material fixed 12th August, 1927, and stained with lactophenol cotton blue; Figure D. Radial section as above showing hyphae in two small vessels. The one to the right shows lower end blocked with wound gum and hyphae embedded in the mass. Fixed 12th August, 1927, stained with lactophenol cotton blue; Figure E. Invaded vessel showing mass of mycelium. Section stained with Delafield's haematoxylin and eosin. Figure F. Section of grape stub four days after inoculation to show spores germinating in vessel. Stained with lactophenol cotton blue. Magnification of A, B, C, D, and E, approximately 350 diameters, that of F, 85 diameters.

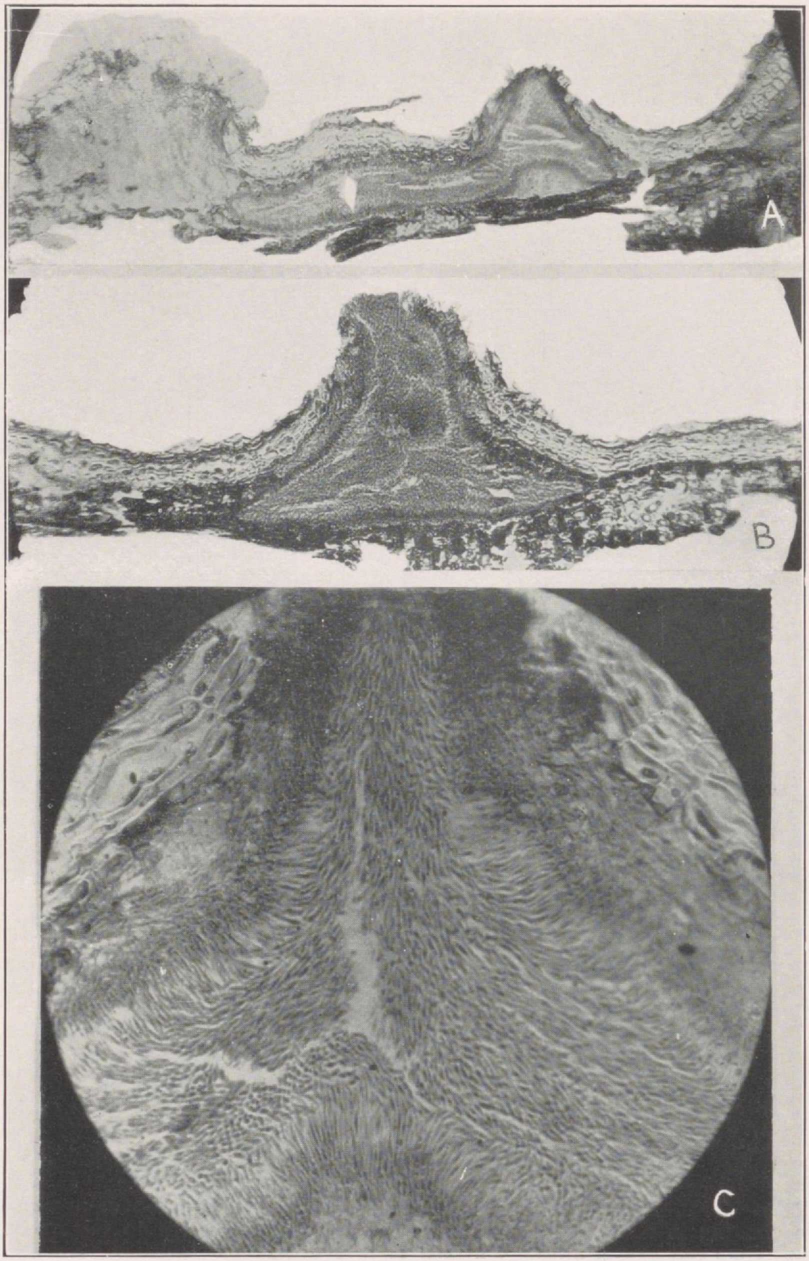

Plate IX. Figure A. Longitudinal section of bark of grape stem, Niagara variety, showing to the right a pycnidium with an extended base breaking through cork layer and to the left a sterile mycelial mass which has broken through; Figure B. Similar section showing pycnidium emitting spores. Most of the emitted mass has been washed away in the preparation. Figure C. Centre of a pycnidium from same material showing attachment of conidiospores and spores being forced towards the ostiole. The material used was fixed in chromacetic acid on the 13th July, 1927. Sections stained in Delafield's haematoxylin and orange G. Magnification in A and B about 75 diameters, in C about 370 diameters.

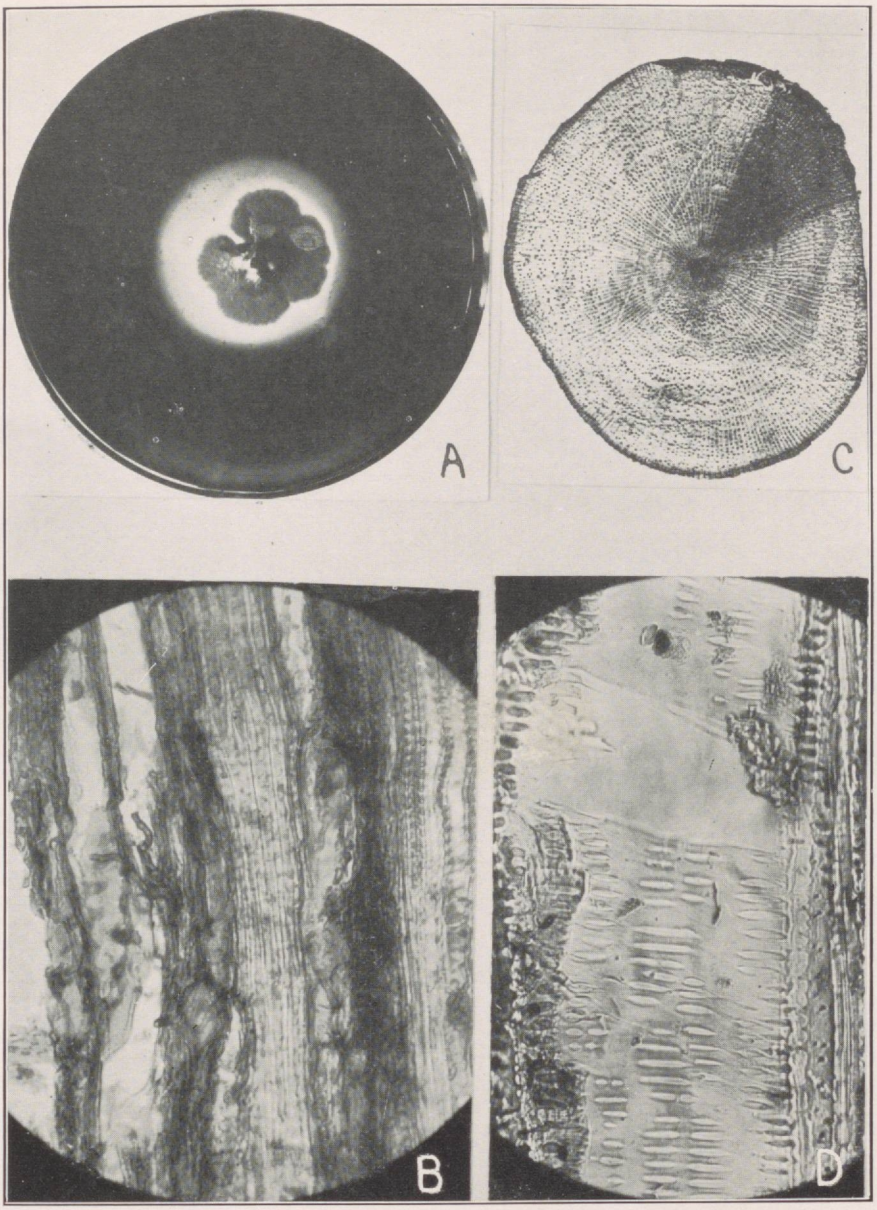

Plate X. Figure A. Young culture of *C. viticola,* Shear, on potato dextrose agar plate. The medium has been flooded with iodine to bring out solution of starch around mycelial mass. Figure B. Radial section through phloem region of a lesion showing blackening and disorganization of cambial region, and penetration of the soft phloem by *C. viticola*. The phloem fibres remain unattacked. Fixed in chromacetic acid 7th February, 1927. Sections stained by Dickson's method. Magnification about 400 diameters. Figure C. Cross section of young grape stem near top to show type of lesion common in early stages of infection extending as a wedge from circumference to centre. Photo slightly enlarged, February, 1927. Figure D. Longitudinal section of branch stub made four days after inoculation with spore suspension of *C. vitiola* and showing spore germinating in vessel. Hand section stained with lactophenol cotton blue. Magnification about 400 diameters. Photo 20th August, 1927.